us: illiterate farming families of simple faith who gave us such hospitality despite their poverty; or missionary doctors who would see in a day the number of outpatients seen here in a week, then sit down and do the hospital accounts, before being interrupted by the need for emergency surgery, an instrumental delivery, or the repair of the generator.

The "BTAf" (been to Africa) degree once mentioned in this journal is a degree in awareness as much as capability. One's reflections on the narrow and privileged world of Western medicine are given cogency, and a glimpse of the realities behind the Oxfam posters gives the necessary perspective to any plans for a future career. For me, 10 weeks in Rwanda gave more than some practical research lessons, a linguistic challenge, and an opportunity for lasting friendships. That short period proved a springboard for my own thinking on medical priorities; it has left an abiding interest in appropriate health care for the world's rural poor—the majority of mankind—and implanted a deep sense of gratitude for all that we in Britain have inherited.

We were kept going from day to day by the enthusiasm of Dr Richard Rowland of Gahini Hospital, whose idea the survey was. We were given valuable advice by the University of London's Tropical Child Health Unit, and financial support by Oxfam, the Medical Research Council, and Edinburgh Medical Missionary Society.

Clinical Topics

Thrombocytosis, circulating platelet aggregates, and neurological dysfunction

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Summary and conclusions

In 12 patients whose neurological dysfunction was associated with thrombocytosis or evidence of abnormal platelet activation, or both, correction of the platelet disorder corresponded with reversal of the neurological symptoms. This suggests that platelet abnormalities may, in certain cases, produce several syndromes of neurological dysfunction, presumably as a result of obstruction of the cerebral microcirculation.

Introduction

There is increasing awareness of the importance of platelets in the pathogenesis of arterial thromboembolic disorders. In cerebrovascular disease the importance of platelets is usually considered in relation to their role in atherogenesis, and few reports have attempted to show that clinical syndromes of neurological dysfunction may relate to specific abnormalities of circulating platelets. Each of the 12 patients in this study had neurological signs and symptoms associated with various abnormalities of platelets and platelet function. Correction of the platelet defect coincided with reversal of the clinical symptoms.

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Patients and methods

Twelve patients (seven male and five female aged 8-72 years) were studied. Nine patients had had transient ischaemic attacks, two had developed neurological signs and symptoms after splenectomy, and one had experienced neurological symptoms after splenic infarction.

The platelet abnormalities included thrombocytosis detected on at least three separate occasions, spontaneous platelet aggregation, and circulating platelet aggregates. Nine patients had raised platelet counts; of these, five had essential thrombocythaemia and four had secondary thrombocytosis. Three patients had circulating platelet aggregates, which in two cases were associated with spontaneous platelet aggregation. This was also detected in blood samples from three of the patients with thrombocytosis (see table).

LABORATORY TESTS

Platelet aggregation responses, including the detection of spontaneous platelet aggregation, were performed by means of a Payton platelet aggregometer.[1] Reversible platelet aggregates were detected by a modification[2] of the method originally described by Wu and Hoak.[3] Low platelet aggregate ratios indicate reversible platelet aggregates. The presence of circulating platelet aggregates, as indicated by this method, was confirmed by inspecting a peripheral blood film. Platelet survival, measured in three patients before and after treatment, was measured by an isotope method using ^{51}Cr.[4]

Results of treatment

Patients with essential thrombocythaemia were treated with both platelet and myelosuppressive drugs. Platelet-suppressive treatment consisted of enteric-coated aspirin, 325 mg, and Persantin (dipyridamole), 100 mg, both thrice daily. All patients improved considerably within a few days of starting treatment. This initial response can almost certainly be attributed to the antiplatelet drugs, since the myelosuppressive agents (radioactive phosphorus, busulphan) require a few weeks to produce an appreciable effect on the platelet count. After correction of the thrombocytosis the patients became symptom-free.

We present details of three patients with secondary thrombocytosis and one with circulating platelet aggregates. Each of the remaining patients received enteric-coated aspirin and Persantin. Within 24

hours of starting treatment circulating platelet aggregates and spontaneous platelet aggregation were no longer evident, and within two weeks all patients were symptom free.

Three of the patients had shortened platelet survival times before treatment (table). After correction of the platelet defect platelet survival, measured in two of these patients, was normal in one (half-life ($t_{\frac{1}{2}}$) 76 h) and greatly improved in the other ($t_{\frac{1}{2}}$ 54 h before treatment and 70 h after it).

Details of patients

Case No	Platelet disorder(s)	Associated or predisposing clinical condition	Neurological features
1	Thrombocytosis	Splenic infarction	Cerebellar signs and symptoms, sensory level T8
2	SPA CPA Reduced platelet survival ($t_{\frac{1}{2}}$ 20 h)	Carotid artery stenosis	Recurrent hemianaesthesia, recurrent vertigo, amaurosis fugax
3	Thrombocytosis	Post-splenectomy	Headaches, confusion, disorientation
4	Thrombocytosis SPA	Post-splenectomy	Grand mal seizures
5	Thrombocytosis	Chronic gastrointestinal haemorrhage	Recurrent hemiparesis and dysphasia
6	Thrombocytosis	Essential thrombocythaemia	Amaurosis fugax, recurrent transient hemiparesis
7	Thrombocytosis CPA	Essential thrombocythaemia	Amaurosis fugax, confusion
8	Thrombocytosis SPA Reduced platelet survival ($t_{\frac{1}{2}}$ 60 h)	Essential thrombocythaemia	Amaurosis fugax, recurrent transient hemianaesthesia
9	Thrombocytosis	Essential thrombocythaemia	Amaurosis fugax, confusion
10	Thrombocytosis CPA	Essential thrombocythaemia	Amaurosis fugax, recurrent vertigo
11	SPA CPA Reduced platelet survival ($t_{\frac{1}{2}}$ 54 h)	TIA No detectable associated or predisposing disorder	Amaurosis fugax
12	CPA	TIA No detectable associated or predisposing disorder	Amaurosis fugax

SPA = Spontaneous platelet aggregation.
CPA = Circulating platelet aggregates.[a]

CASE 1

A 60-year-old man with essential thrombocythaemia was admitted complaining of difficulty in walking and standing and was unable to keep his balance. The previous night he had retired to bed clinically well, but during the night he experienced nausea and severe left upper quadrant abdominal pain that persisted for a few hours. He showed evidence of gross cerebellar insufficiency and had a sensory level at T8 with sparing of the sacral segments indicating an intrinsic cord lesion. His platelet count was extremely high (1200 $\times$ 10^9/l) and the peripheral blood film indicated hyposplenism. He was thought to have suffered infarction of his spleen during the night, which had caused the considerable increase in his peripheral platelet count. Both the cerebellar insufficiency and the intrinsic cord lesion were considered to be ischaemic in origin and to have been caused by vascular occlusion by platelets.

The next day thrombopheresis was carried out using an Aminco cell separator (kindly performed by Dr R J Sokol, Blood Transfusion Service, Sheffield), and this effectively reduced his platelet count to 500 $\times 10^9$/l. He was also given enteric-coated aspirin, 325 mg, and Persantin, 100 mg, both thrice daily. Within five days he could stand and walk unaided, with no evidence of any residual neurological abnormality.

CASE 2

A 40-year-old man first presented with spontaneous, painless loss of vision in the right eye. Angiographic studies showed bilateral carotid artery stenosis. Vascular surgery was attempted on the left carotid artery but the results were considered unsatisfactory, and the vessel remained impalpable. Six months later the patient complained of recurrent transient blurring of vision in the left eye, transient right hemianaesthesia, and two episodes of vertigo. He also had intermittent claudication and was unable to walk more than 150 metres without resting. Clinical examination showed no new features. All laboratory investigations were normal apart from a slightly raised platelet count (415 $\times 10^9$/l). Platelet aggregates were noted on the peripheral blood film. Reversible aggregates were confirmed by a low platelet aggregation ratio (0·5). Platelet function tests showed spontaneous platelet aggregation, and increased platelet turnover with shortened platelet survival ($t_{\frac{1}{2}}$ 20 h, normal 72-97 h) was shown by isotope studies using ^{51}Cr.

The patient was given enteric-coated aspirin, 325 mg, and Persantin, 100 mg, both thrice daily. Within 24 hours the platelet aggregates were no longer detectable, and one month later he was no longer experiencing transient cerebral ischaemic attacks or amaurosis fugax. No episodes of transient cerebral ischaemia have occurred during the past three years. Platelet survival was measured after three months of platelet-suppressive treatment and was found to be normal ($t_{\frac{1}{2}}$ 76 h).

CASE 3

A 24-year-old housewife presented in 1973 with generalised convulsions associated with hypoglycaemia. An insulinoma was diagnosed, and the whole of the pancreas, together with her spleen, was removed. Her postoperative course was uneventful until on the 11th day she became confused and aggressive, and complained of photophobia and severe headache. She then became disorientated and required continuous nursing attention. The cerebral symptoms continued for the next four days. Her pulse and blood pressure were unchanged, and she remained apyrexial. Her blood sugar content, which was monitored several times each day, remained throughout between 8·2 and 21 mmol/l. Plasma sodium, potassium, calcium, magnesium, and bicarbonate concentrations were all normal; the urea concentration was low. The onset of her cerebral signs and symptoms coincided with a rise in platelet count to 900 $\times 10^9$/l on the 11th day after splenectomy. Platelet function tests, performed for the first time on the 14th day after splenectomy (that is, three days after the cerebral symptoms appeared), showed spontaneous platelet aggregation and enhanced aggregation to adenosine diphosphate and collagen. She was treated with aspirin, Persantin, and subcutaneous heparin. Spontaneous platelet aggregation was no longer evident when platelet function tests were repeated a few hours after starting treatment. Her cerebral symptoms disappeared the next day, and she has remained well.

CASE 4

A previously healthy 8-year-old boy underwent splenectomy for steroid-resistant idiopathic thrombocytopenic purpura. The postoperative recovery was at first uneventful. The platelet count, which had been 20 $\times 10^9$/l preoperatively, rose progressively until by the ninth postoperative day it was 600 $\times 10^9$/l. On this day he was well and apyrexial, but during sleep he had the first fit of his life, which was a generalised seizure. Three hours later he had a second grand mal convulsion, again affecting all limbs. He had no focal neurological signs, and examination of the fundus oculi was normal. Temperature, pulse, and blood pressure were all normal as was the cerebrospinal fluid, and cultures were sterile.

Platelet function tests, performed six hours after his first fit, showed spontaneous platelet aggregation. We thought that his fits were possibly caused by cerebral vascular occlusion resulting from thrombocytosis associated with spontaneous platelet aggregation. He was given soluble aspirin, 300 mg twice daily, and Persantin, 50 mg four times daily. Enhanced platelet aggregation, measured 24 hours later, was no longer evident. Results of an examination of the central nervous system were normal, and there were no further fits. He was maintained on platelet-suppressive treatment until his platelet count returned to normal, when treatment was discontinued. He has remained well with no treatment.

Discussion

Although platelets are recognised as important factors in the pathogenesis of arterial thrombotic disease, relatively few reports show that platelet abnormalities may produce clinical syndromes of vascular insufficiency.

In this study neurological dysfunction occurred in association with thrombocytosis, spontaneous platelet aggregation, and circulating platelet aggregates. A causal relation is suggested by the observation that correction of the platelet defect was

accompanied by the disappearance of the abnormal clinical signs and symptoms. In two patients evidence of improved platelet survival was also obtained. The conclusion that cerebrovascular insufficiency may occur in association with thrombocytosis irrespective of the underlying mechanism producing it is of some importance. Although thrombosis is known to occur in patients with essential thrombocythaemia, it is usually considered to affect either large vessels or the smaller vessels in the foot producing peripheral ischaemia.[5] It is not always appreciated that various neurological effects, such as amaurosis fugax, recurrent hemiparesis, vertigo, and confusion, may also occur in these patients. Similar clinical findings also occurred in patients with thrombocytosis associated with chronic haemorrhage or after splenectomy or splenic infarction. This confirms the observations of Haselager and Vreeken[6] that thromboembolic complications are not confined to myeloproliferative syndromes but may also occur in association with secondary thrombocytosis.

Spontaneous platelet aggregation and circulating platelet aggregates were observed in blood samples from six patients. In three patients both of these abnormalities were present. Although the significance of spontaneous platelet aggregation remains unknown, it appears to be a phenomenon associated with thromboembolism that is confined to groups of patients with thromboembolic disease but is rarely observed in blood samples from normal individuals. In the context of neurological dysfunction, ten Cate[7] observed a high incidence of spontaneous platelet aggregation in patients with various forms of cerebrovascular disease, including transient ischaemic attacks; and this appeared to correlate with an increased sensitivity of the patients' platelets to aggregating agents.

Evidence for circulating platelet aggregates has been noted in patients with transient cerebral ischaemia,[8] and although it is not certain whether these are cause or effect of the cerebral ischaemia their continued presence in the circulation will certainly predispose to further vascular obstruction. One of our patients (case 11—see table) had circulating platelet aggregates without clinical or radiological evidence of vascular abnormality, while another (case 2) had grossly abnormal carotid vessels; probably the platelet aggregates in the latter were platelet emboli from the abnormal vessel wall. Regardless of the mechanisms producing these aggregates both patients responded identically to the platelet-suppressive drugs aspirin and Persantin in that circulating platelet aggregates were no longer evident, platelet survival was considerably improved, and the clinical signs and symptoms were abolished.

The results of this study provide evidence that certain abnormalities of platelets and of platelet function may produce syndromes of vascular insufficiency affecting the nervous system. Considerable clinical improvement may be achieved by platelet-suppressive drugs and, where appropriate, reduction of the platelet count.

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Clinical medical officers in the child health service

Report of the Joint Paediatric Committee of the Royal Colleges of Physicians and the British Paediatric Association*

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The tripartite structure of the health services prior to 1974—general practice, hospital practice, and the local authority medical service—affected paediatrics particularly, as the majority of doctors employed by local authorities were engaged in child health. The 1974 National Health Service Act provided an opportunity for establishing an integrated child health service but unfortunately did not make proper provision for the future employment and training of doctors formerly employed by local authorities who wished to continue as child health clinicians. This failure weakened the child health services, prejudiced effective clinical integration, and resulted in considerable injustice to this group of doctors.

Both the Brotherston (1973)[1] and the Court (1976)[2] Reports envisaged the health care of children as primary, largely general practice based, on the one hand and secondary, largely hospital based, on the other. Within primary care there are, however, paediatric tasks for which general practitioners may be untrained or which they do not wish to undertake. These include developmental assessment, advising schools and teachers about specific medical problems in education, routine school medical examinations, multidisciplinary assessment, health education, etc. Although secondary child health care is currently largely focused on hospitals, there is need for a wider deployment of secondary child health care beyond hospital confines and for certain elements of secondary child health care operating outside hospitals to relate more closely to hospitals. "Hospital paediatrics" and "community paediatrics" are not two separate disciplines. Each requires the understanding of childhood diseases, which are the same wherever they occur. If childhood diseases such as cerebral palsy, spina bifida, epilepsy, mental retardation, congenital heart disease, asthma, impairment of the special senses, malignancy, cystic fibrosis, chronic renal disease, dyslexia, diabetes, psychiatric disturbances, and many

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